Article

Epidemiology of toe tip necrosis syndrome (TTNS) of North American feedlot cattle

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Abstract — Toe Tip Necrosis Syndrome (TTNS) is predominantly a hind limb lameness of feedlot cattle that develops early in the feeding period. Retrospective analyses of feedlot health records were conducted in order to describe the epidemiology of the disease at the level of the individual animal, lot, and feedyard. Analysis of 1904 lots (cohorts of > 100 head) of cattle, from 48 feedyards, found that TTNS occurred sporadically, but clustered by both lots and feedyards. Only 3.8% of lots had ≥ 1 case of TTNS; however, 26.4% of these lots were associated with 1 feedyard. Analysis of 702 cases of TTNS found that the disease clusters early in the feeding period; the mean (median; range) number of days on feed at death was 42.3 d (27.0 d; 4 to 302 d). The disease occurred in all months of the year and affected calves, yearlings, steers, and heifers. It was equivocal as to whether the source of the animals was associated with how quickly they died of TTNS in the feedyard.

Résumé — Épidémiologie du syndrome de la nécrose du bout des doigts (SNBD) du bétail dans les parcs d'engraissement d'Amérique du Nord. Le syndrome de la nécrose du bout des doigts (SNBD) est une boiterie des membres postérieurs du bétail des parcs d'engraissement qui se développe de 1 à 4 semaines après l'arrivée au parc d'engraissement. Des analyses rétrospectives des dossiers de santé des parcs d'engraissement ont été réalisées afin de décrire l'épidémiologie de la maladie au niveau de l'animal individuel, du lot d'animaux et du parc d'engraissement. Une analyse de 1904 lots (cohortes de > 100 têtes) de bétail, provenant de 48 parcs d'engraissement, a constaté que le SNBD se produisait sporadiquement, mais qu'il était regroupé selon les lots et les parcs d'engraissement. Seulement 3,8 % des lots avaient ≥ 1 cas de SNBD; cependant, 26,4 % de ces lots étaient associés à 1 parc d'engraissement. Une deuxième analyse des 702 cas de SNBD a confirmé que la maladie se regroupe au début de la période d'engraissement; le nombre moyen de jours (médiane; écart) d'engraissement à la mort était de 42,3 jours (27,0 jours; de 4 à 302 jours). La maladie se produisait durant tous les mois de l'année et touchait les veaux, les animaux d'un an, les bouvillons et les génisses. Il était équivoque à savoir si la source des animaux était associée à la rapidité d'une mort causée par SNBD dans le parc d'engraissement.

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Introduction

Lameness is a significant disease of feedlot cattle. A retrospective study of ~1.84 million animal health records from American feedlots found that lameness accounted for 16% of treatments, 5% of deaths, and 70% of animals sent prematurely to slaughter were lame (1). A similar retrospective study of Canadian feedlot cattle found that bovine respiratory disease and lameness accounted for 42% and 40% of all treatments, respectively (2). Respondents to a more recent US survey of feedlot nutritionists, veterinarians, and managers, estimated the mean (median) incidence of lameness in feedlots to be ~3.8% (2.0%). Interdigital necrobacillosis (footrot) was considered the most common cause of lameness, followed by injury (35%), and "toe abscesses" (10%) (3). While toe abscesses were identified as a common cause of lameness, there is a paucity of information regarding the epidemiology of this disease in beef cattle.

While toe abscesses of feedlot cattle are distinct from sole ulcers and abscesses of dairy cattle (4), one of the best descriptions of toe abscesses of feedlot cattle involved a cohort of Jersey heifers in New Zealand (5). This initial report was followed by 2 reports relating to outbreaks of toe abscesses of beef cattle in American feedlots (6,7). More recently, researchers, again from New Zealand, reported on dairy heifers that developed hoof lesions similar to what is seen in feedlot beef cattle (8). Some of the first observations regarding the epidemiology of the disease

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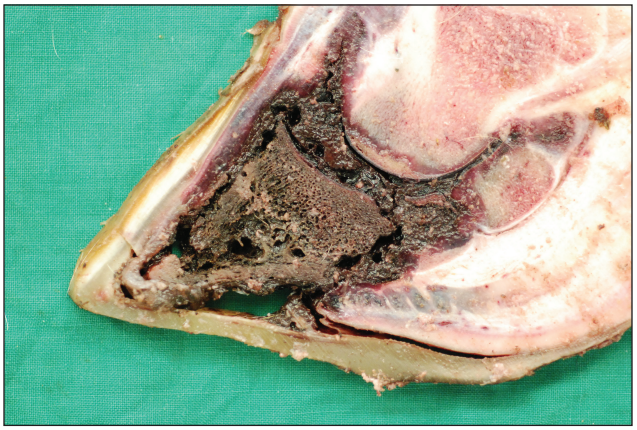

Figure 1. Photograph of a sectioned claw taken from an animal with toe tip necrosis syndrome.

and potential risk factors originate from these early publications. Specifically, affected cattle develop lameness shortly after being shipped and/or handled at a processing facility. Furthermore, the disease was associated with fractious or agitated animals and if the animals were exposed to wet and/or abrasive flooring conditions.

The cause of toe abscesses in feedlot cattle is a matter of speculation; however, the "abrasion theory" is perhaps the most widely accepted explanation. This theory postulates that excessive wear of the solar horn leads to separation along the apical portion of the white line, allowing for a secondary bacterial infection. These infections may penetrate the corium and progress to P3 osteitis, P2 osteomyelitis, tendonitis, tenosynovitis, cellulitis, and in some cases septicemia that leads to an embolic event that culminates in death. If this theory is correct, then events that lead to excessive wear along the apical white line should be considered risk factors for the disease.

Although the disease is common to North American feedlots, no uniform nomenclature has been adopted to describe the disease. The condition has many different monikers: toe abscess, toe ulcer, *apicus necrotica*, apical white line disease, toe necrosis, toe tip necrosis, "P3" necrosis, and apical pedal bone necrosis. We prefer the term toe tip necrosis syndrome (TTNS) (9) because it describes the primary pathological finding (toe tip necrosis) (10) and it encompasses the sequelae commonly associated with the condition (6,7).

The objective of the study was to describe the epidemiology of TTNS at the level of the individual animal, the lot, and the feedyard.

Materials and methods

The over-arching objective of the study was to describe the epidemiology of TTNS at the level of the individual animal, the lot, and the feed yard (feedlot). Feedlot Health Management Services (FHMS), a large multi-person feedlot consulting practice in western Canada (Okotoks, Alberta), provided the source data.

Lot and feedyard

The first objective was to describe the occurrence of TTNS by lot and feedyard, in which a lot was defined as a cohort of cattle purchased from ≥ 1 source, but grouped for financial reporting reasons. Data were restricted to lots of > 100 head, feedyards located in western Canada, and the 2012 calendar year. Due to client confidentiality concerns, the number of TTNS cases per lot was provided as prevalence data (percent).

Table 1. Fatal cases of toe tip necrosis syndrome by source of cattle and age class

Source (N = 696)	Calf	Yearling
Auction (*n* = 545)	271 (49.7%)	274 (50.3%)
Backgrounded (*n* = 69)	18 (26.1%)	51 (73.9%)
Grass (*n* = 53)	1 (1.9%)	52 (98.1%)
Ranch (*n* = 29)	22 (75.9%)	7 (24.1%)
Totals	*n* = 312 (44.8%)	*n* = 384 (55.2%)

The case definition for TTNS was finding evidence of necrosis of the apex of the toe along with pathology of the 3rd phalangeal bone (P3) at the time of postmortem examination. This diagnosis was made after making a sagittal section of the claws with an axe or saw. While concurrent disease processes may have been present, if 1 or more claws had lesions that satisfied the case definition, and the lesion was deemed to be the primary reason for euthanasia or cause of natural death, then the animal was recorded as a TTNS case.

Figure 1 is a photograph of a sectioned claw taken from a TTNS case. The P3 bone is necrotic and pathological processes involve P2 and associated soft tissues. White line separation, which is pathognomonic for TTNS, was not evident in the photograph because the hoof was sectioned either medial or lateral to the point of separation.

Individual animal

The FHMS' animal health database was queried for confirmed fatal cases of TTNS reported between January 1, 2008 and December 31, 2012, inclusive. These individual animal data originated from feedyards in western Canada and the western United States. Individual animal health records provided the following data: arrival weight; arrival date; source of procurement (auction, grass, backgrounded, ranch); gender (heifer or steer); age class (calf or yearling); number of days on feed (DOF) at 1st treatment for TTNS; number of DOF at death; date of death; whether the animal was found dead or was euthanized; location of TTNS lesion [front or hind claw(s)]; and estimated/actual weight at death. Regarding 1st treatment for TTNS, a putative diagnosis of TTNS lameness was based upon the animal exhibiting lower limb lameness, but having no observable signs of swelling or evidence of other disease processes such as foot rot or traumatic injury.

Statistical analyses

Data were compiled in a commercial spreadsheet software program (Microsoft Excel, v.15; Microsoft Corporation, Redmond, Washington, USA) and then exported to a statistical software program (STATA, ver 14; StataCorp LP, College Station, Texas, USA). Descriptive statistics were generated for each variable, but only statistically significant findings were reported. The Kruskal-Wallis and the median test were used to assess for differences in the number of DOF until 1st treatment and until

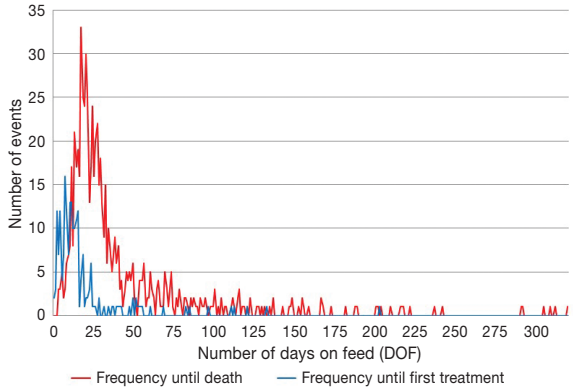

Figure 2. Epidemic curves for number of days on feed until first treatment for toe tip necrosis syndrome lameness and until death.

death by source, age class, and gender. The Mann-Whitney U-test assessed for differences in the number of DOF between the treated and nontreated cattle. Logistic regression was used to estimate the odds of the treated and nontreated cattle having to be euthanized for lameness. Kaplan-Meier (K-M) survival analyses, using the Wilcoxon test statistic, assessed for the overall equality of the survivor curves (survival functions) over time. Specifically, K-M survivor analyses compared the number of DOF until 1st treatment and DOF until death by source, age class, and gender. The level of significance for all statistical tests was set at $P < 0.05$ (two-tailed).

Results

Lot and feedyard

Lot prevalence data were compiled from 1904 lots of cattle, ranging in size from 100 to 5443 head, representing 616 831 head of cattle from 48 feedyards. Most lots (96.2%; 1832/1904) reported no cases of TTNS; 72 lots had ≥ 1 case of TTNS. Nineteen of 48 (39.6%) feedyards reported having 1 or more lots with a TTNS case. One feedyard accounted for 26.4% (19/72) of all the affected lots and 33.3% (19/57) of lots entering this same feedyard had ≥ 1 TTNS cases.

The prevalence of TTNS cases in the 72 affected lots ranged from 0.50% to 1.22%. The feedyard with the highest prevalence in a single lot (1.22%) only reported having 4 of 110 (3.6%) lots having TTNS cases.

Individual animal

A query of FHMS' database returned 702 recorded TTNS cases, 6 of which had 1 or more missing data points. There was a steady decline in the number of TTNS cases recorded in each successive year: 2008, $n = 208$; 2009, $n = 212$; 2010, $n = 155$; 2011, $n = 97$; and 2012, $n = 30$. There were no differences across the years (2008 to 2012) with respect to either the number of DOF at the time of 1st treatment for suspected TTNS lameness ($P = 0.72$) nor the number of DOF at death due to TTNS ($P = 0.42$); therefore, all cases were analyzed as 1 dataset.

Table 1 is a cross tabulation of the cases by source and age class. Most (78.3%; 545/696) cases were auction market-derived cattle. Across all sources, the cases were nearly evenly split between calves (44.8%; 312/696) and yearlings (55.2%; 384/696); most of the calves (77.3%; 242/313) and yearlings (65.0%; 249/383) were steers.

The epidemic curves for DOF until 1st treatment for TTNS lameness and until death followed a similar pattern with the peak number of treatments and deaths occurring early in the feeding period (Figure 2).

Table 2 provides the breakdown of DOF to 1st treatment and to death by source, age class, and gender. The overall mean (median) number of DOF at 1st treatment was 18.9 d (12.0 d), which was unrelated to source ($P = 0.80$), age class ($P = 0.98$), and gender ($P = 0.59$). A third (29.9%; 210/702) of the animals had a history of having been treated for TTNS lameness. The timing of 1st treatments started on the day of arrival (0 DOF) and continued until 203 DOF. Thirty-seven (17.6%; 37/210) animals were given antimicrobial therapy within 5 DOF, 81.1% (30/37) of which were dead within 15 d of treatment. Furthermore, 64.9% (24/37) of these early onset animals were euthanized for lameness.

The overall mean (median) number of DOF at death was 42.3 d (27.0 d); range 4 to 302 DOF. Table 2 provides a breakdown of the number of DOF at the time of death by source, age class, and gender. Thirteen animals with TTNS were dead within 7 DOF, 8 (61.5%) of which were euthanized. Most cattle (77.9%) died within 50 DOF and overall 75.2% (528/702) of animals with TTNS were euthanized versus being found dead.

The median number of DOF at death between the treated (25 DOF) and nontreated cattle (28 DOF) was not different ($P = 0.18$); however, treated cattle were twice as likely to be euthanized as were the nontreated cattle (OR = 1.8; 95% CI = 1.20 – 2.71; $P < 0.01$).

Kaplan-Meier (K-M) survivor analysis was used to determine if there were differences in the overall survivor functions. That is, rather than just looking at differences in the mean or median DOF until death, the K-M test compared the probabilities of the occurrence of an event (death) over time. Figure 3 is the graphical representation of the survivor functions. All 702 animals were alive at the start of the feeding period (DOF = 0), and each descending step represents the death of ≥ 1 animal(s) in a given time interval. Kaplan-Meier univariate analysis found differences in the survivor functions with respect to the number of DOF until death by source ($P = 0.04$) and the number of DOF by age class ($P = 0.04$), when examined over the time period from Day 0 to 302. Cattle that were backgrounded had a greater overall survival time than did the cattle from the other 3 sources. Similarly, the calves survived for a longer period than did the yearlings. The 2 variables (source and age class) were then analyzed together by controlling (stratifying) for age class (calf *versus* yearling). Overall, there were differences in the survivor functions by source of cattle ($P = 0.02$). While differences existed in the survivor functions between 0 to 302 DOF, there were no differences in the survivor functions by source for the period 0 to 100 DOF ($P = 0.59$). This is significant because 91.2% (640/702) of the cattle died within the first 100 DOF.

Animals died or were euthanized in all months of the year; however, 67.9% (477/702) of deaths occurred from September to December.

Table 2. The mean (median), standard deviation (SD), and range in number of days on feed (DOF) at time of 1st treatment for toe tip necrosis syndrome and for death, stratified by the source, age class, and gender

	DOF at 1st treatment	DOF at death
Source (N = 696)		
Auction (*n* = 545)	20.4 (12.0); SD ± 27.5; 1 − 203	40.6 (27.0); SD ± 40.6; 4 − 309
Backgrounded (*n* = 69)	17.4 (12.0); SD ± 18.9; 0 − 85	69.0 (38.0); SD ± 75.6; 7 − 320
Grass (*n* = 53)	13.6 (13.0); SD ± 8.3; 1 − 37	32.4 (27.0); SD ± 22.2; 7 − 121
Ranch (*n* = 29)	16.2 (16.4); SD ± 5.1; 8 − 21	44.1 (25.0); SD ± 53.1; 11 − 222
Age class (N = 702)		
Calf (*n* = 318)	20.8 (12.0); SD ± 29. 8; 1 − 203	49.5 (28.0); SD ± 57.1; 5 − 320
Yearling (*n* = 384)	17.5 (12.0); SD ± 19.5; 0 − 121	37.1 (26.0); SD ± 32.0; 4 − 217
Gender (N = 696)		
Steer (*n* = 491)	18.8 (12.0); SD ± 25.2; 0 − 203	43.6 (27.0); SD ± 48.6; 4 − 320
Heifer (*n* = 205)	19.1 (13.0); SD ± 22.2; 2 − 121	39.5 (28.0); SD ± 35.9; 8 − 217

The location of the TTNS lesion was recorded in 35 cases: 1 animal (2.9%) had a TTNS lesion on a fore foot; 2 animals (5.7%) had lesions on both a hind and a fore foot; 7 animals (20.0%) had lesions in both hind feet; and 25 animals (71.4%) had a lesion in 1 hind foot.

Discussion

The objective of this study was not to prove or disprove the "abrasion theory;" however, many of the findings can be explained within the context of this theory.

The disease occurred sporadically, but clustered at the level of the lot and feedyard. Less than 4% of the lots had ≥ 1 cases of fatal TTNS, with the highest recorded prevalence in a single lot being 1.22%. Significantly, outbreak of TTNS occurred in a feedyard in which the disease was seldom reported; only 4 of 110 lots of cattle had 1 or more cases of fatal TTNS. This suggests that the risk factors for the disease may be associated with the incoming lots of cattle. Conversely, ~25% of all the affected lots were associated with a single feedyard, which infers that feedyard-specific risk factors may also have been present. The sporadic nature of the disease coupled with the clustering by lots and feedyards suggests that the risk factors for TTNS may be ephemeral. That is, the risk factors may not always be present in time and space.

The treatment and necropsy data confirmed previous reports that TTNS clusters early in the feeding period (5–7). Of the 210 animals that received antimicrobial therapy for a presumptive diagnosis of TTNS, 50% were ≤ 12 DOF. Furthermore, confirmed cases of TTNS occurred as early as 4 DOF. This clustering of cases early in the feeding period indicates that the animals were exposed to risk factors prior to, on the day of, or within a few days of arrival at the feedyard. However, treatments and deaths continued for many months into the feeding period. In these cases it is unlikely that TTNS was initiated early in the feeding period only to have clinical signs manifest many months later. Rather, a more likely scenario is that the late cases represent newly developing cases, which implies that the same constellation of risk factors may exist at multiple time points in the feeding period. For example, mustering cattle for reimplanting and/or revaccination may replicate the same conditions that animals encounter before or shortly after arrival at the feedyard.

Of the animals administered antimicrobial therapy for a putative diagnosis of TTNS, 25% were dead within 6 d. This rapid progression suggests that in some cases the disease may not be responsive to antimicrobial therapy or that the treatments were given too late. It was also of interest that 70% of animals died without having received antimicrobial therapy. These findings are not an indictment of the feedyard personnel. Rather, TTNS occurs as a lower limb lameness without obvious signs of swelling and hence cases may be misdiagnosed as traumatic injuries, which typically do not warrant antimicrobial therapy. These data underscore that the disease can be difficult to accurately diagnose clinically, unless time and effort are made to restrain the animal and perform a close inspection of the foot. This is logistically challenging and hence we suspect that early cases of TTNS are probably being misdiagnosed and underreported. This is of concern because a diagnosis of TTNS changes how the animals should be treated (i.e., aggressive debridement, claw amputation, antimicrobial therapy) and also changes the prognosis. Once the P3 bone is affected, then aggressive therapy such as claw amputation is often required to salvage the animal. If this course of action is not taken, then euthanasia must be considered.

One of the presumptive risk factors for TTNS is handling cattle on abrasive surfaces such as stamped, etched, or wet concrete. Mason et al (8) described an outbreak of hoof lesions in dairy heifers, which included white line separation and toe abscesses. Unlike feedyard cattle, these dairy heifers did not develop lesions until 2 mo after arrival at the dairy. The foot lesions were attributed to the heifers being fed on a wet and coarse concrete feed pad and being commingled with adult cows. Commingling of the heifers and cows was thought to have agitated the heifers, which may have contributed to the outbreak. This is of interest because hyper-excitability has been suggested as a possible risk factor for TTNS in feedyard cattle (7,11). Conceivably, mustering fractious cattle on abrasive flooring may result in excessive wear along the apical region of the white line, leading to white line separation. If the texture (abrasiveness) of the flooring is an important risk factor, then the accumulation of manure, snow, and ice may alter the surface textures, which could explain the apparent ephemeral nature of the disease.

While exposure to abrasive flooring during transport, at auctions, or in feedyards may account for the clustering of cases

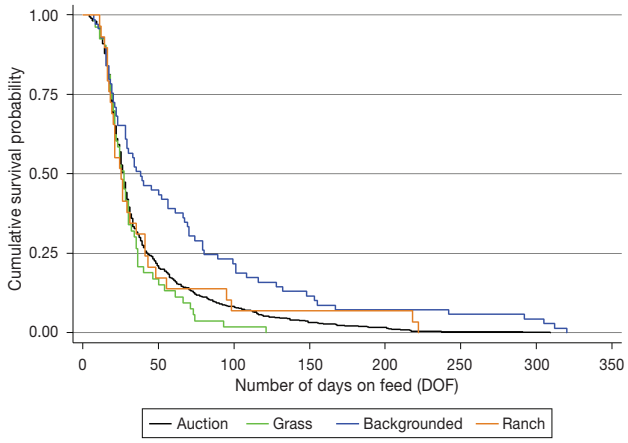

Figure 3. Graphical representation of the survivor functions.

early in the feeding period, veterinarians have also speculated that standing for prolonged periods such as during transport may impede blood perfusion to the corium, resulting in white line separation. There is no evidence to support this conjecture; however, disturbances in the micro-vascularization of the corium may contribute to the pathogenesis of laminitis and other claw lesions (12–14). Therefore, prolonged standing should be considered as a potential risk factor for TTNS.

The biomechanical properties of the hoof must also be considered as a potential factor in the development of the disease. Hoof horn tissue is very dynamic with respect to its ability to hydrate and dehydrate (15), which in turn influences its hardness and elasticity (15–17). This has clinical significance because increasing moisture content of solar horn has been associated with wear and thinning of the soles and indirectly with white line separation (18). This is salient because wet environmental conditions have been posited as a risk factor for TTNS (7). If this is correct, then the disease may be clustering within cohorts of animals coming off wet pastures, or being exposed to wet environmental conditions at auctions and feedyards. Furthermore, these conditions would change with time, helping to explain the sporadic nature of the disease.

Other factors that influence hoof hardness are the pigmentation of the horn tissue and the animal's nutritional status. Hoof hardness has been shown to increase with the level of pigmentation (15) and micronutrients are important for maintaining the integrity of the hoof horn (15,19–21). Given the myriad of factors that could potentially influence the rate of wear to the soles, it is perhaps not surprising that the disease occurs sporadically but clusters by lot and feedyard.

Most of the TTNS lesions were found in the hind feet; however, lesions occasionally occurred in the front feet. The predilection for TTNS to occur in the hind feet was consistent with previous reports (5–7). However, consistent with most previous reports, Smith and Brodersen (22) reported TTNS-like lesions that primarily involved front feet. This latter report underscores that veterinarians and feedyard crews need to be cognizant that TTNS may also account for forelimb lameness.

Most cases involved auction-derived cattle, steers, and yearlings. However, these findings probably reflect the proportion of auction-derived cattle, steers, and yearlings fed over the 5-year study period. Similarly, most cases occurred in the months of September to December, which coincides with when cattle typically enter the feedyard. Therefore, there was probably no seasonal effect *per se*; rather, the seasonality was confounded by when cattle enter the feedyards. The overriding conclusion from the individual animal data is that TTNS can develop in calves and yearlings; steers and heifers; and in all months of the year.

The Kaplan-Meier analysis provided a graphical representation of when the animals died after entering the feedyard. It must be stressed that without a control group the graphs only convey the timing of the deaths in the feedyard; no conclusions can be drawn as to whether source was a risk factor for the occurrence of the disease. While the backgrounded cattle survived longer in the feeding period than did the cattle from the other 3 sources, this finding may be spurious and therefore must be interpreted with caution. There was no difference in the 4 survivor functions in the first 100 DOF, a period when 90% of all cattle died. Therefore, if source had an effect on the speed of the progression of the disease, then it was largely masked by other factors in the first 100 DOF. However, the divergence of the survival curve for the backgrounded cattle from the other groups is intriguing and deserves additional study.

There were a number of limitations to this study. Only lots of $>$ 100 head were included so as to avoid a small number of cases skewing the lot prevalence data. Larger lots were also chosen because producers and auction markets frequently sort less thrifty cattle into smaller lots, which could have introduced other biases. Only 1 y of lot prevalence data were analyzed, which may have influenced the results. All the records were obtained from clients of FHMS and their recommendations for when and how to treat lame cattle may or may not represent what is occurring across the North American feedlot industry. It also needs to be emphasized that some of the clustering of cases in time and space may be related to a detection bias, with some feedlot personnel being more capable and vigilant when it comes to detecting TTNS cases.

Despite the limitations, this was the first study dedicated to describing the epidemiology of TTNS in North American feedyard cattle. The data confirm anecdotal reports that the disease is sporadic, but clusters in time, by lot, and by feedyard. Clustering early in the feeding period suggests that TTNS is initiated prior to, during, or shortly after arrival at the feedyard. Clustering by lot may be related to the cohorts sharing common attributes such as temperament, environmental (wet) conditions, nutritional status, and claw pigmentation (breed). Conversely, clustering by feedyard portends the potential for feedyard-specific factors such as facility design, flooring, animal handling, and the ability of the feedyard crews to identify and treat cases early in the course of the disease. Lastly, it was salient that the number of TTNS cases had decreased significantly over the 5-year time period. This reduction may be related to changes in how lame cattle are being identified and/or treated. A well-designed prospective study is needed to determine the true prevalence of the disease as well as provide greater clarity on potential risk factors. CVJ

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Erratum

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Abnormal changes in both mandibular salivary glands in a dog: Non-mineral radiopaque sialoliths

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